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ABSTRACT

Bulimia represents a major health problem in the United States, especially in high schools, colleges, and universities. This paper reviews literature on the definition, etiology, and therapeutic approaches for bulimia. The literature points to potential physiological bases for the disorder since some researchers reported similarities between it and the physiological aspects of major affective disorders. Some of this research is supported by the relative efficacy of treatment models for both conditions with include antidepressants, especially the tricyclics. But much of the literature is flawed, with some researchers relying only on self-report data to determine compliance with medication. Improved methodology would allow use of validated paper-and-pencil measures with the examination of the bulimic's blood plasma level of the drug. Research using these techniques plus self-reports, clinical and behavioral observations, and chemical and urine analyses may he determine the efficacy of antidepressant, psychotherapeutic, or behavior management strategies on bulimic behavior. The research surveyed here suggests that when clinicians are either assessing or treating bulimic clients, they should also investigate for any major affective disorder. Once the presence of other mental disorders is detected, clinicians can begin to design more effective treatment modalities for bulimia. Contains 43 references. (RJM)

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INTERVENTION WITH ADOLESCENTS: THE IDENTIFICATION OF BULIMIA NERVOSA, AND A POSSIBLE TREATMENT MODALITY

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OVERVIEW

Bulimia and anorexia nervosa are major health problems in the United States, especially in high schools, colleges, and universities. Published studies on the prevalence of bulimia, depending on the operational definition, include 26% to 79% of women assessed who report binge eating (Mitchell & Eckert, 1987).

Some authors (e.g. Mintz & Betz, 1988) have noted that fully developed cases of bulimia do not appear to be as prevalent on college campuses as many researchers believe. Mintz and Betz (1988) found that only 20 out of 643 (3%) of the subjects in their study could be classified as being bulimic; however, only 211 out of 643 (33%) subjects reported "normal" eating habits. Drewnowski, Hopkins and Kessler (1988), using a national sample of 1,007 male and female college and university students in the United States and using Diagnostic and Statistical Manual of Mental Disorders (Third Edition-Revised) (DSM-III-R; American Psychiatric Association, 1987) criteria, found that 1% of the women and 0.2% of the men in their sample were classified as bulimic. The DSM-III-R (American Psychiatric Association, 1987) states that in a study of college freshman, 4.5% of the females and 0.4% of the males had a history of bulimia. When a criterion

of at least one binge and purge episode per week was applied, Shisslak, Crago, Neal, and Swain (1987) examined various studies and found that the prevalence rate of bulimia in high school and college students varied from 1% to 3%. Halmi, Falk, and Schwartz (1981) found that of 355 college students they examined, 13% reported experiencing all of the major symptoms of bulimia.

Diagnosis is often difficult due to reliance on self-reports and the often clandestine nature of bulimics' behaviors; in addition, the treatment picture with this population is even more obscure (Mitchell & Eckert, 1987; Walsh, Gladis, Roose, Stewart, Stetner & Glassman, 1988). Traditional long-term psychotherapy has often been difficult to maintain with bulimics; this may be due to a lack of client motivation, a lack of success, or to other unknown factors (Mitchell & Eckert, 1987). Fortunately, recent advances in pharmacological therapy, particularly in the area of antidepressant medications, have increased the efficacy of some treatment models. Pharmacological therapy seems to be an alternative worthy of consideration; it requires less intrinsic motivation than psychotherapy; it may be used to control the bulimic behavior when used in conjunction with other therapies and, in a sense, can be more cost-effective. Some studies have

suggested that the use of antidepressant medications alone can be successful in the treatment of bulimia (e.g. Hughes et al., 1986; Walsh et al., 1988). Some studies have also hypothesized that a physiological basis exists for bulimia (e.g. Kaplan & Woodside, 1987). Finally, some studies have attempted to link bulimia with affective disorders, usually major depression (Kaplan & Woodside, 1987; Mitchell & Eckert, 1987).

The Operational Definition of Bulimia

Most studies of bulimia use the DSM-III-R's or the Diagnostic and Statistical Manual of Mental Disorders (Third Edition)'s (DSM-III; American Psychiatric Association, 1987; 1980) definition of bulimia. The DSM-III-R criteria which must be met in order for someone to be diagnosed as bulimic include:

1. Recurrent episodes of binge eating (rapid consumption of a large amount of food in a discrete period of time. Eating binges may be planned. The food that is consumed during these binges may have a high caloric content, a sweet taste, and a texture that facilitates rapid eating. The food is usually eaten as inconspicuously as possible, or secretly. A binge is usually terminated by abdominal discomfort, sleep, social interruption, or induced vomiting.

The food eaten during a binge may be gobbled down rather rapidly, with little chewing;

2. A feeling of lack of control over eating behavior during the eating binges;

3. Self-induced vomiting, use of laxatives or diuretics, strict dieting or fasting, or vigorous exercise in order to prevent weight gain. Vomiting decreases the physical pain of abdominal distention, allowing either continued eating or termination of the binge, and often reduces post-binge anguish. In some cases vomiting itself may be desired, so that the person may binge in order to vomit or will vomit after eating a small amount of food. Although eating binges may be pleasurable, disparaging self-criticism and a depressed mood often follow;

4. Persistent overconcern with body weight and shape. Bulimics invariably exhibit great concern about their weight and make repeated attempts to control it by dieting, vomiting, or the use of diuretics. Frequent weight fluctuations due to alternating binges and fasts are common. Bulimics may feel that their lives are dominated by conflicts surrounding eating;

5. The subject must have had, on the average, a minimum of two binge eating episodes a week for at least three months in order to qualify for the diagnosis of bulimia nervosa. Some bulimics may be slightly underweight and some may be overweight, but most people with bulimia are within a normal weight range. A depressed mood that might be part of a Depressive Disorder is often seen. Some bulimics may have Psychoactive Substance Abuse or Dependence, most often involving sedatives, amphetamines, cocaine or alcohol (American Psychiatric Association, 1987, pp. 67-69).

The parents of bulimic individuals are often obese (American Psychiatric Association, 1987). A higher than expected frequency of Major Depression in first degree biologic relatives of bulimic people has been reported in several studies (e.g. Geracioti & Liddle, 1988; Keck, Pope, Hudson, McElroy, Yurgelun-Todd & Hundert, 1990). The course of the illness is chronic and intermittent, lasting a period of many years (American Psychiatric Association, 1987, p. 68).

Etiology

Physiological Mechanisms

Hypothalamus

Gandour (1984) and Herzog (1986) proposed that bulimia may be caused by a hypothalamic dysfunction, but Gandour (1984) concluded that the notion of a dysfunctional hypothalamus in bulimics should remain at the hypothetical stage. Irregular eating patterns may, in some way, alter neurotransmitter levels to change the release of hypothalamic hormones. Kaplan and Woodside (1987) noted that the hypothalamic-pituitary function is indeed disturbed in people who have anorexia nervosa or bulimia; the disturbance may be of either the unique contributions of the pituitary or the hypothalamus or an interaction of the two. However, at least some of these disturbances are generally mediated by mechanisms that are secondary to the effects of disordered eating. For example, abnormalities in thermoregulation and in gonadotropic hormone levels relate directly to weight loss and are seen in other states of emaciation, regardless of the cause. These abnormalities, like amenorrhoea, may reflect the effects of weight loss, disordered eating, or other factors that are not known. Hsu (1990) noted that amenorrhoea can occur in response to nonspecific emotional stress, malnutrition, and weight loss. In a

recent study of normal college-age women, two to five percent reported having amenorrhoea and ten percent reported having menstrual abnormalities (Hsu, 1990).

Neurotransmitters

Other physiological mechanisms involving the hypothalamus may have neurotransmitter bases. Kaplan and Woodside (1987) also support the view, found most often in the animal literature, that neurotransmitters are directly implicated in the control of feeding behavior. Serotonin inhibits feeding by inducing satiety; norepinephrine stimulates feeding through its activity in the medial hypothalamus and paraventricular nucleus. Norepinephrine can also inhibit feeding through its action on the lateral hypothalamus. Both of these neurotransmitters may be affected by the availability of certain nutrients; for example, serotonin appears to be regulated by the availability of tryptophan. Infusion of norepinephrine into the medial hypothalamus causes a specific preference for carbohydrates (Hsu, 1990) and seems to be produced when food is ingested (Kaplan & Woodside, 1987). Kaplan and Woodside (1987) also noted that serotonin may be involved in the regulation of mood, pain, sleep, appetite, and in the control of the hypothalamic and pituitary hormones. The key here is that all of these functions have been found to be disturbed in people

with eating disorders.

Mitchell and Eckert (1987) noted that, in animal studies, the release of norepinephrine into the hypothalamus stimulated the animals to eat, even if the animal was sated from previous exposure to food. The amount of carbohydrates ingested also increased. Carbohydrate consumption produces an increase in the precursor for serotonin production. As serotonin increases, the rising level produces the opposite effect by decreasing eating behavior, specifically decreasing the desire to ingest carbohydrates (Mitchell & Eckert, 1987). Results of human studies showed that serotonin dysfunction and disregulation may occur in bulimics, thus possibly leading to the disinhibition of carbohydrate intake. Davis, Freeman and Garner (1988) reported that bulimics tended to binge more on grains and cereals during binging episodes, one of the diagnostic criteria of the DSM-III-R (American Psychiatric Association, 1987). Neurotransmitters, such as norepinephrine and serotonin, may be involved in the biological bases of eating disorders, in the behavioral symptomatology as well as in the dynamic chemical state of the individual.

Hormones

Hormone levels have also been shown to vary between groups of bulimic and normal individuals.

Geracioti and Liddle (1988) found that the hormone cholecystokinin was significantly impaired in bulimic patients. This was determined by comparing the bulimics' blood plasma levels of cholecystokinin with those blood plasma levels of normal women.

Cholecystokinin, located in the brain and in the gut, has been found to be involved in satiety regulation in rats, sheep, monkeys, and in humans (Bennett & McMartin, 1979; Geracioti & Liddle, 1988). Cholecystokinin, an amino acid polypeptide hormone secreted by the membrane of the upper intestine after eating, is also secreted by the hypothalamus. Cholecystokinin stimulates gallbladder contractions and pancreatic secretions and may impact on gut motility. That expansion or contraction of the gut, signaling satiety or hunger, often seems to be impaired in bulimics evidencing clinical symptoms such as dyspepsia (indigestion) and early satiety (Kaplan & Woodside, 1987). Geracioti and Liddle (1988) concluded that bulimics do not have normal satiety and that they also have impaired secretion of cholecystokinin in response to a meal. A key suggestion made by these authors is that both of these abnormalities may be improved by tricyclic antidepressants (a class of antidepressant drugs), although the mechanism by which they may augment cholecystokinin secretion is unknown.

Cortisols

Musisi and Garfinkel (1985) reported that cortisol secretion may be disturbed in bulimics. Abnormal results were noted in the dexamethasone suppression test (DST) in bulimics of normal weight. The DST's major purpose was to test an individual for endogenous depression and results may be due to depressive symptoms themselves, to abnormal eating patterns, or to a biologically underweight state even though normal weight is maintained. As a point of comparison, abnormal DST results may be related to weight loss and have been seen in nondepressed patients who had lost weight for other reasons (Fichter, Pirke & Holsboer, 1986).

As the literature demonstrates, no definite conclusions regarding the physiology of bulimia can be stated; however, a number of hypotheses are suggested. If a hypothalamic dysfunction is involved in bulimia, the specifics of that dysfunction and the nature of its involvement are not yet clear. Serotonin and norepinephrine are shown in relation with the feeding-satiety process and may be dysfunctional in people who manifest bulimia and other eating disorders. It can also be hypothesized that serotonin and norepinephrine are related to the function of the hypothalamus and may reflect abnormalities. There is literature to support

this view yet the origin of dysfunctional effects and the nature of interaction still remains unsubstantiated.

More importantly, the literature hypothesizes that the satiety function is impaired in bulimics and that serotonin and norepinephrine levels are also impaired. Included in this hypothesis is the notion that cholecystokinin levels are also impaired (Geracioti & Liddle, 1988). However, these physiological manifestations may be the results of disorders in eating, not the etiology. In effect, these studies demonstrate that a relationship appears to exist between a person's physiology and bulimia, but the actual cause of bulimia has yet to be identified.

Bulimia and Affective Disorders

There appear to be common physiological and affective characteristics (such as disturbed sleep, low self-esteem, feelings of sadness, and general lethargy) in the symptomatology of bulimia and affective disorders; consequently these affective symptoms may be related to bulimia (Bazire, 1988; Hudson & Pope, 1990; Siegel, 1989; Swift, Andrews & Barklage, 1986).

Familial Incidences

Studies have demonstrated that some bulimics have family histories of major affective disorders (e.g. Geracioti & Liddle, 1988; Jampala, 1985). Major

depression has also been identified as a specific diagnosis among immediate family members (Geracioti & Liddle, 1988; Logue, Crowe & Bean, 1989; Siegel, 1989). Keck et al. (1990) compared 119 bulimic women with nonpsychiatric controls and found that the bulimic women differed significantly from the controls in their prevalence of personal and familial major affective disorders. Keck et al. (1990) concluded that their data support the hypothesis that a relationship exists between bulimia and major affective disorders.

Hudson, Pope, Yurgelun-Todd, Jonas and Frankenburg (1987), in structured diagnostic interviews, found that bulimics had high lifetime rates of major affective disorders and anxiety disorders. Pope, Hudson and Yurgelun-Todd (1989) compared the scores of 45 bulimics, 21 people in treatment for major depression and 27 normal controls on the Hamilton Rating Scale for Depression and found that the bulimic and depressed subjects were virtually indistinguishable on all items. Both of these groups were easily distinguished from the controls, thus adding support to the hypothesis that major depression and bulimia may be associated.

Hsu (1990) noted that about one-third of those patients who had an eating disorder (anorexia nervosa or bulimia) also had major depression. However, he noted that current evidence did not support the view

that eating disorders are necessarily related to affective disorders or are variants of them.

Hypothesizing that bulimia may share a pathophysiological abnormality with major depression and with panic disorder, Hudson and Pope (1990) reviewed literature that examined all treatment studies using four classifications of antidepressants: tricyclics, monoamine oxidase inhibitors, serotonin uptake inhibitors, and atypical antidepressants such as trazedone. Hudson and Pope (1990) evaluated disorders that consistently responded to antidepressants and that were not classified as affective disorders. They discovered three placebo-controlled studies for each drug that demonstrated the efficacy of using desipramine, imipramine and phenelzine in the treatment of bulimia, with little or no counterevidence. They also discovered ten placebo-controlled studies that demonstrated the effectiveness of treating panic disorder with imipramine. Hudson and Pope (1990) concluded that the treatment-response model they developed identified bulimia and panic disorder as possible components of the overall affective disorder spectrum, which typically is seen as only consisting of major depression.

Antidepressant Medications

The efficacy of pharmacological treatment for chronic and severe depression has been substantiated over time; it has been a relatively brief period that these same pharmacological agents have been used in treatment for bulimia. Patterns of positive response may add support to a common physiological base for some depression and eating disorders.

As with most of the drugs used to treat psychiatric problems, the mechanisms of action of tricyclic antidepressants are not completely understood (Baldessarini, 1985). The literature poses several hypotheses about how these drugs may work. Baldessarini (1985) states that, in addition to the acute sedative effects of the tricyclics, they also serve to enhance the action of norepinephrine and may block the effects of tyramine, a neurotransmitter. These observations have led to support for the amine hypothesis of affective disorders, suggesting that major depression is associated with a relative lack of activity of certain amine neurotransmitters in the brain, most probably norepinephrine. Other amines such as acetylcholine and serotonin may also be involved in major depression. As noted earlier, both norepinephrine and serotonin have been implicated in physiological studies of bulimics and therefore have

been hypothesized as being dysfunctional in physical substrates of bulimia (Kaplan & Woodside, 1987; Mitchell & Eckert, 1987).

Bulimia and Antidepressant Medication Treatment

Geracioti and Liddle (1988) raise key issues regarding the treatment of bulimia nervosa with tricyclic antidepressants. Research has shown some interesting results concerning the efficacy of treating bulimia with antidepressants. Imipramine, a tricyclic antidepressant, has been shown to increase the available amount of the monoamine neurotransmitters norepinephrine or serotonin (or both) in the central nervous system. The reuptake of norepinephrine and serotonin into the postsynaptic neurons is blocked by the tricyclics, thus allowing serotonin and norepinephrine to accumulate and prolonging their action (Barnhart, 1986; Gilman, Rall, Nies & Taylor, 1990; Springhouse Corporation, 1989). The tricyclics need an average of two to four weeks for effective levels, most often the latter to attain maximum effectiveness. Primary indications have been for various conditions, such as major depression, enuresis (especially using imipramine), panic disorder, and bulimia. These drugs may be effective in the treatment of organic affective disorders, obsessive-compulsive disorder, and posttraumatic stress disorder (Hyman &

Arana, 1987).

Pope and Hudson (1982) performed one of the first studies on the treatment of bulimia with the antidepressant imipramine. Although their sample size was small (N=8), they found that 75% of those subjects who were treated with imipramine showed a significant decrease in their binging behavior, lasting from two to seven months. However, Pope and Hudson (1982) did not have a control group in their study and could not rule out that the results were caused by experimental bias, imipramine therapy, or some extraneous variable. Hudson, Pope, and Jonas (1983) found supporting data in a subsequent study where bulimic symptoms were significantly reduced in treatment with imipramine.

Mitchell, Pyle, Eckert, Hatsukami, Pomeroy and Zimmerman (1990) reported that the improvement experienced by bulimic outpatients was superior for those who were in intensive group psychotherapy when compared to those who were taking imipramine alone. The addition of imipramine to the intensive group psychotherapy component did not significantly affect the improvement of bulimic symptoms when compared to the addition of a placebo, but it did result in greater improvement in the symptoms of depression and anxiety. All three active treatment phases (imipramine alone, imipramine with psychotherapy and placebo with

psychotherapy) resulted in significant reductions in target eating behaviors (binging and purging) and a significant improvement in mood (Mitchell et al., 1990).

Desipramine is another tricyclic antidepressant whose mechanisms of action, primary indications, and time of effective therapeutic onset are similar to imipramine; however the adverse anticholinergic side effects (dry mouth, constipation, urinary hesitancy, etc.) and sedation are less severe than imipramine's (Gilman et al., 1990; Hyman & Arana, 1987). In a six week trial of desipramine with bulimics, Hughes et al. (1986) measured serum desipramine levels during the fifth week of the study in order to see how many of their subjects had achieved blood plasma levels within the therapeutic range for desipramine. Their results demonstrated that bulimics who were treated with desipramine showed an 87% binge decrease rate. The results are interpreted with caution, however, due to two factors: a decrease in sample size caused by attrition and the use of self-report measures (created by the authors and called a "global clinical ratings scale" and a "bulimia symptom rating scale") for reporting data which were of questionable validity.

Barlow, Blouin, Blouin and Perez (1988) assessed 47 normal weight bulimics who were treated with

desipramine during a 15 week, double-blind crossover experiment. Twenty-four subjects completed the 15 week trial, with desipramine being significantly more effective than a placebo in reducing the frequency of weekly binging and regurgitation.

Subsequently, Blouin, Blouin, Perez and Barlow (1989) performed a randomized double-blind crossover trial of desipramine on 24 normal weight bulimics, again over a period of 15 weeks. Desipramine was significantly more effective than a placebo in reducing the frequency of weekly binging and vomiting. Seven subjects responded to desipramine by reducing their binging, seven were classified as borderline responders, and the other ten were non-responders. Interestingly, responders had a higher frequency of purge episodes than the non-responders.

Monoamine oxidase inhibitors (MAOI's), such as phenelzine, promote the action of monoamine neurotransmitters by inhibiting the enzyme monoamine oxidase in the central nervous system, peripheral nervous system and nonnervous tissues such as the liver and gut. The time required for effective levels to build for MAOI's is from one to four weeks, approximately equivalent to the onset time for the tricyclics. The primary indication for these drugs is for the treatment of major affective disorders and for

panic disorders (Barnhart, 1986; Gilman et al., 1990; Hyman & Arana, 1987; Springhouse Corporation, 1989). These drugs have been used with some success in the treatment of bulimia. However, since complex, severe and/or unpredictable interactions between MAOI's and other drugs and food-derived amines may occur, using them is difficult and may be potentially hazardous (Gilman et al., 1990).

Pope, Hudson and Jonas (1983) reported that MAOI's may be superior to other antidepressants in the treatment of bulimia. Of 65 patients who were treated in their clinic for bulimia, 17 out of 26 (65%) bulimic patients showed a marked response or remission with MAOI's, even though many had responded poorly to tricyclics or to trazedone. Walsh et al. (1988) noted that phenelzine was successful in 64% of their bulimic clients in reducing binge frequency. However, MAOI's have some serious side effects and require the user to be on a tyramine-free diet. This dietary restriction may be prohibitively difficult to maintain with bulimics and may carry serious health consequences when breached (Walsh et al., 1988).

Chemically different from the tricyclics or MAOI's, the antidepressant trazedone strongly inhibits serotonin reuptake in the brain. The time of therapeutic onset is similar to the tricyclics and to

MAOI's, and its primary indication is for the treatment of major affective disorders. One advantage of trazedone is that its anticholinergic side effects are minimal; however, it retains sedative effects and may still produce drowsiness and dizziness (Barnhart, 1986; Gilman et al., 1990; Hyman & Arana, 1987; Springhouse Corporation, 1989).

Solyom, Solyom and Ledwidge (1989) used trazedone with 13 bulimics in an open label, flexible dose study. Three subjects dropped out before the fourth week; however, in those subjects who remained, the number of binging and vomiting episodes was significantly decreased. The number of episodes was reduced to zero in four subjects. The mean weight of the subjects was virtually unchanged from pre- to post-treatment: 58.5 kg to 57.3 kg. Solyom et al. (1989) concluded that this lack of weight gain is an advantage of using trazedone over the other antidepressants since the fear of becoming fat is part of the bulimic's pathology.

Fenfluramine is an anorectic drug that stimulates the ventromedial nucleus of the hypothalamus, may affect the sympathetic and parasympathetic nervous systems and may have serotonin depleting properties. In addition, it may alter the hunger and/or satiety mechanisms. It is primarily indicated for use by obese individuals as an appetite suppressant and as a weight

reducer (Gilman et al., 1990; Springhouse Corporation, 1989). Blouin, Blouin, Perez, Bushnik, Zuro and Mulder (1988) ran a 15 week, double-blind, placebo controlled crossover trial of 22 normal weight bulimic subjects. 12 subjects were randomly assigned to a group receiving fenfluramine and the other 10 subjects received desipramine. Blouin et al.'s (1988) results demonstrated that both drugs had beneficial effects on the frequency of binging and regurgitation, although more subjects responded positively to fenfluramine than to desipramine. Both drugs were also effective in reducing the psychological symptoms of bulimia such as the urge to binge.

Russell, Checkley, Feldman and Eisler (1988) performed a 12 week double-blind, placebo controlled trial of fenfluramine with 42 bulimics. Forty percent (17 out of 42) of the subjects dropped out halfway through the 12 weeks. Among the noncompleters, those on fenfluramine had reportedly experienced relief of their bulimic symptoms; the authors hypothesized that the persistence of depressive symptoms and of the features of bulimia may have contributed to the high dropout rate. Those taking fenfluramine did not evidence weight loss or serious side effects, but fenfluramine by itself did not significantly benefit some of the subjects who had severe bulimia. Due to

this high dropout rate, the authors advised caution when interpreting their results, and they called for more research into treating bulimia with fenfluramine.

Based upon the literature, using antidepressant medications in the treatment of bulimia would seem to be a logical course of treatment, even if we are not sure how or why these medications work or if bulimia and major affective disorders are somehow related. However, there were limitations in the earlier research.

For example, Brotman, Herzog and Hamburg (1988) discovered that three out of seven of their clients who received at least one trial of antidepressants showed a significant decrease in their binging behavior. However, even though Brotman et al. (1988) found positive treatment effects, they also had a small sample, thus hindering their study's external validity. Other studies as well (e.g. Hughes et al., 1986; Russell et al., 1988) also suffered from sample sizes which grew even smaller due to attrition.

Conclusion

Many questions remain for future research of the mechanisms related to the etiology and treatment of bulimia. The literature points to potential physiological bases and similarities to the physiological aspects of major affective disorders.

These hypotheses are supported by the relative efficacy of treatment models for both conditions which include antidepressant agents, especially the tricyclics (e.g. Hughes et al., 1986). However, much of the literature is flawed. For example, some researchers have relied only on self-report data to determine compliance with medication. Improved methodology would allow use of validated paper-and-pencil measures with the examination of the bulimic's blood plasma levels of the drug, like the Hughes et al. (1986) study. A study using these techniques plus self-reports, clinical and behavioral observations, and chemical and urine analyses may help detect the efficacy of antidepressant, psychotherapeutic, or behavior management strategies on bulimic behavior. The value in measuring blood levels, which yields vital information including such data as compliance as well as levels of cholesterol, potassium, etc., cannot be underestimated.

Although psychopharmacological research provides some evidence for positive and efficacious treatment avenues, other problems may arise. Many antidepressants have side effects, some of which may be unpleasant (Baldessarini, 1985; Hyman & Arana, 1987). One way to counteract this is to use a drug that has low sedating effects and low anticholinergic effects.

Hyman and Arana (1987) recommend desipramine, among other drugs, in order to achieve these results. MAOI's also appear to be effective but the fact that they would require the bulimic to remain on a tyramine-free diet makes their usage unwise. The concept of treating bulimia nervosa with tricyclic antidepressants remains controversial for many reasons, not the least of which is that the front-line usage of these drugs is for depression, not for treating bulimia (Gilman et al., 1990; Springhouse Corporation, 1989). Regardless, investigations into drug therapy as an effective treatment modality for bulimia may add to the support that drug therapy may be used as a viable treatment option and that bulimia may somehow be linked with affective disorder.

The results of the presented studies suggest that when clinicians are either assessing or treating bulimic clients, they should also investigate, and treat if present, any major affective disorder. The clinician will have to decide which disorder warrants primary attention. Once the presence of other mental disorders (in addition to bulimia) is detected, clinicians can begin to design more effective treatment modalities for bulimia.

The causal factor(s) of bulimia are still unknown. Regardless of what these factors may be, it remains

unclear whether bulimia contributes to the manifestation of depression or whether depression contributes to the manifestation of bulimia (Mitchell, 1990).

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